

more functions of the liver may be disturbed without affecting the other hepatic functions. One objection to the study of the function of any organ as an index of disease of that organ is that it is perhaps possible for the healthy portion of the diseased organ to compensate and assume the work of the whole gland. In such a condition, of course, the functional capacity of the organ may be normal and would be no index of the pathological changes in that organ. Under these circumstances only marked destructive changes would leave their impress on the functional sufficiency of the organ.

ETHEREAL SULPHATE ELIMINATION BEFORE AND AFTER THYMOL ADMINISTRATION.

| Case No. | Diagnosis. | Total sulphur, gms. | | Etheral sulphate sulphur, gms. | | Etheral sulphate sulphur, per cent. | |
|----------|---------------------|---------------------|---------------|--------------------------------|---------------|-------------------------------------|---------------|
| | | Before thymol. | After thymol. | Before thymol. | After thymol. | Before thymol. | After thymol. |
| 1 | Normal | 2.0375 | 2.1295 | 0.2893 | 0.5646 | 14.2 | 26.8 |
| 2 | Gastritis | 1.9428 | 1.7427 | 0.1457 | 0.3380 | 7.5 | 19.4 |
| 3 | Fracture | 2.7467 | 2.5527 | 0.3131 | 0.6024 | 11.4 | 23.6 |
| 4 | Congestion of liver | 0.9852 | 0.0734 | 0.1753 | 0.7069 | 17.8 | 26.8 |
| 5 | Congestion of liver | 1.7345 | 1.6982 | 0.2480 | 0.3610 | 14.3 | 21.2 |
| 6 | Gall-stones | 2.7628 | 2.8075 | 0.7597 | 1.0303 | 27.5 | 36.7 |
| 7 | Gall-stones | 3.0042 | 2.6826 | 0.3965 | 0.8474 | 13.2 | 29.4 |
| 8 | Cholecystitis | 2.7807 | 2.6437 | 0.4866 | 0.7428 | 17.5 | 28.1 |
| 9 | Atrophic cirrhosis | 2.2328 | 2.3029 | 0.2791 | 0.3400 | 12.5 | 15.2 |
| 10 | Tumor of liver | 1.9492 | 1.8757 | 0.1637 | 0.3676 | 8.4 | 19.6 |
| 11 | Cancer of liver | 2.7526 | 2.6278 | 0.6083 | 0.6648 | 22.1 | 25.3 |
| 12 | Syphilis of liver | 2.8104 | 2.9075 | 0.3990 | 0.5437 | 14.2 | 18.7 |

The results that we have obtained in the cases cited in the accompanying table are very encouraging. The work is now in progress. We are collecting comparative data as to the positive incidence of this test and other tests of liver sufficiency in the same case of hepatic disease.

SOME CLINICAL AND EXPERIMENTAL OBSERVATIONS ON GASTRIC ACIDITY USE OF THE GAS-CHAIN METHOD.

BY GOLDER L. MCWHORTER, M.D., PH.D.,
CHICAGO.

(From the Department of Surgery, University of Minnesota.)

In order to determine the effect produced by chronic inflammatory and altered physiological changes in the gastro-intestinal tract, I have undertaken the study of the gastric secretion in such conditions by means of the exact gas-chain method of determining the acidity and have plotted curves, comparing the results with those gained by

the titration method. Previous to this time acidity curves have been made clinically only by the titration method. My estimations were made from specimens collected at short intervals after test meals in clinical cases and in animal experiments. In order to obtain pure juice in the latter experiments Pawlow pouches were made and estimations of secretion carried on before and after the production of lesions or altered physiological conditions. In such surgical conditions there may be an increased acidity in the stomach contents; in these observations I have attempted to determine whether such an increase is due to an actual increased acidity in the gastric juice.

The apparatus used in the gas-chain method of determining the hydrogen ion concentration was put at my disposal by J. F. McClendon.¹ This apparatus consists of a hydrogen electrode, a calomel electrode and a potentiometer for measuring electromotive force. The principle of the gas-chain method is well described by Sedgwick.² As to the colorimetric determination of the hydrogen ion concentration the results so far, when applied to gastric contents containing varying amounts of protein and salts, have proved inaccurate and unavailable for following changes in reaction during digestion.

A solution is acid in reaction when it contains an excess of hydrogen over hydroxyl ions, neutral when they are in equal numbers and alkaline when hydroxyl ions predominate. Pure distilled water dissociates into hydrogen and hydroxyl ions, the extent of the dissociation being such that in one liter of water at 22° C. there is approximately $\frac{1}{1000000}$ gram of hydrogen ions; that is, concentration of the hydrogen ions is $\frac{1}{1000000}$ normal (atomic weight of hydrogen as 1). The shorter method of representing so many figures is usually adopted, the logarithmic notation: thus, $\frac{1}{1000000}$ H. Acid = 10^{-7} or simply pH 7. Since there is one hydroxyl ion formed for each hydrogen ion the concentration of the hydroxyl ions must also equal pH 7.

The measurement of the pure gastric juice has been made by Menten,³ and found to vary from pH = 0.92 to 1.58, highest in the appetite juice and lowest in the secretion in the empty stomach. Michaelis and Davidsohn⁴ state that the average acidity of the stomach contents after an Ewald test meal means 0.028 to 0.0015; hyperacidity, 0.011 to 0.088; hypoacidity, 0.000,0041 to 0.000,0001. McClendon,⁵ after normal meals, found a rise in acidity for two or

¹ Hydrogen and Hydroxyl Ion Concentration in Physiology and Medicine, Med. Rev. of Rev., 1916, xxii, 333.

² Hydrogen Ion Concentration of the Gastric and Duodenal Contents in Children, Tr. Am. Pediat. Soc., May, 1915.

³ Acidity of Undiluted Normal Gastric Juice from a Case of Human Gastric Fistula, Jour. Biol. Chem., 1915, xxii, 341-343.

⁴ Die Bedeutung und die Messung der Magensaftacidität, Ztschr. f. Exper. Path. u. Therap., 1910-1911, viii, 398.

⁵ Acidity Curves in the Stomachs and Duodenums of Adults and Infants Plotted with the Aid of Improved Methods of Measuring Hydrogen Ion Concentration, Am. Jour. Physiol., 1914, xxxv, 191-199.

three hours, when it remained constant until the food had left the stomach. Carlson⁶ believes that the view of Pawlow⁷ that gastric juice is secreted at uniform and constant acidity is true for man only in regard to the appetite, digestive and hunger juice secreted at a fairly high rate, and has observed that the normal gastric mucosa is capable of secreting a juice of submaximal acidity. Hardt⁸ after experimental production of gastric and duodenal ulcers found no increased acidity. Grey⁹ after cholecystostomy found no change in the acidity of the gastric secretions.

Pawlow believes that the slower rate of secretion may give a chance for the HCl acid to be partly neutralized by the mucus in the stomach. Boldyreff¹⁰ has shown the entrance of the intestinal contents to be the most important factor in neutralization of the stomach contents.

For the purpose of comparison of acidity estimations a standard test meal was adopted which consisted of two slices of bread with crusts removed and two glasses of distilled water. In all estimations the Rehfuss¹¹ tube was used for collecting specimens. Collections were made every fifteen minutes from the time the test meal was eaten until no more material could be aspirated. Unfiltered gastric juice was used in the estimations, though at times it was strained through coarse gauze. In order to compare the actual acidity to the acidity obtained by the usual titration methods, samples of the juice were titrated against a $\frac{N}{50}$ KOH solution, using dimethyl amino azobenzene and phenolphthalein as indicators. I have found that the value of these indicators used clinically in determining free and total acidity by titration with an alkali is in many cases parallel, though not equivalent, to the actual acidity as determined by the gas-chain method (Fig. 1). Much discussion has arisen as regards the value of the titration methods; consequently, I have made observations by both methods, and have plotted, also, corresponding curves to compare the values of the dimethyl amino azobenzene acidity to the actual acidity. Fig. 1 shows the curves running fairly constantly parallel throughout; however, the acidity as determined by the "indicator method" is uniformly higher than the actual acidity. Fig. 2 shows the plate for determining the hydrogen ion concentration from the dimethyl and phenolphthalein acidity as

⁶ The Secretion of Gastric Juice in Man, Am. Jour. Physiol., 1915, xxxvii, 51. A Note on the Chemistry of Normal Human Gastric Juice, Am. Jour. Physiol., 1915, xxxviii, 248.

⁷ The Work of the Digestive Glands, 1910, translated by Thompson.

⁸ The Secretion of Gastric Juice in Cases of Gastric and Duodenal Ulcers, Am. Jour. Physiol., 1916, xl, 314.

⁹ Jour. Exper. Med., 1916, xxiii, 15.

¹⁰ Self-regulation of Acidity of Gastric Contents and Real Acidity of Gastric Juice, Quarterly Jour. Exper. Physiol., 1914, vii, 1-12.

¹¹ A New Method of Gastric Testing, with a Description of a Method for Fractional Testing of Gastric Juice, AM. JOUR. MED. SC., 1914, cxlvii, 848.

determined with the above indicators. This assumes the acidity to be in terms of pure HCl acid, which dissociates, as shown by the

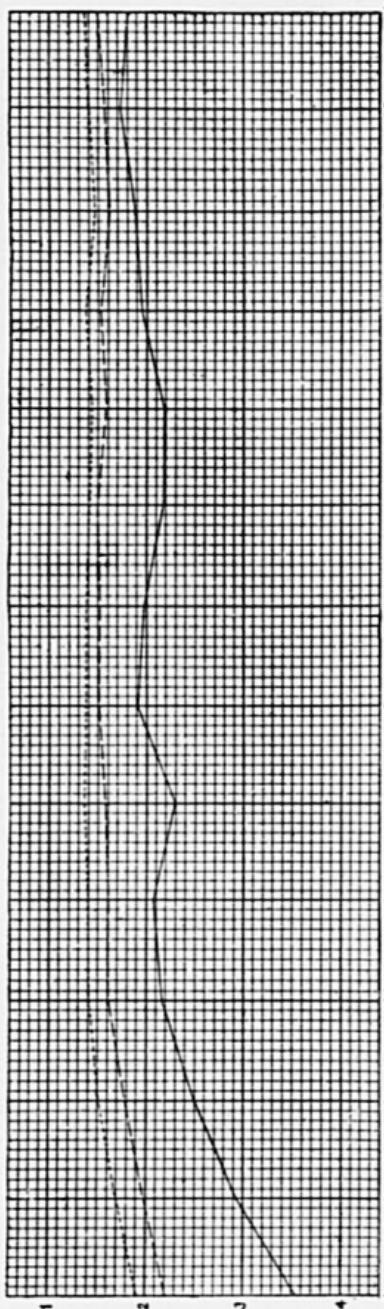


FIG. 1.—The lower solid line is a composite of 32 acidity curves in groups I and II by the use of the gas-chain method of determining the pH. The broken line represents a composite curve of the pH derived from the dimethyl amino azobenzene acidity, assuming it to be pure HCl. The dotted line in the same way represents the phenolphthalein acidity. The estimations were made from specimens collected at fifteen-minute intervals.

curve. Fraenkel¹² found the Congo-red acidity to be almost constantly parallel to the actual acidity as determined by the gas-chain method, and states that the phenolphthalein acidity does not show the free HCl acid even in the pure gastric juice.

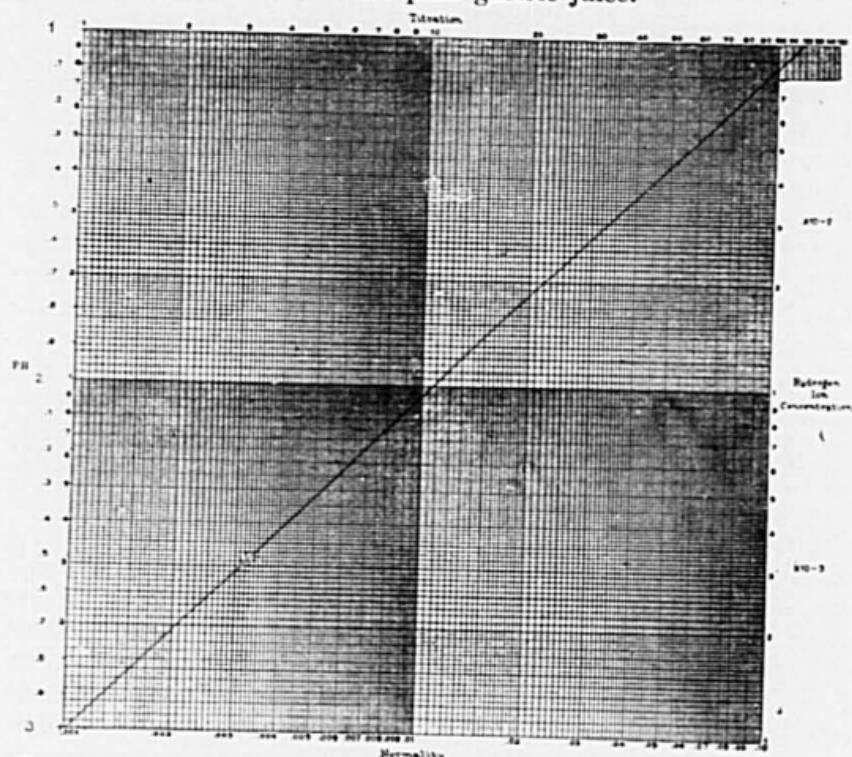


FIG. 2.—By this chart the degree of dissociation of HCl at different degrees of strength may be determined and either the hydrogen ion concentration or the pH estimated. The curve continued from 120 c.c. N/10 HCl = pH 1 would show the dissociation of HCl at higher concentrations. 130 c.c. N/10 HCl = pH 0.996; 140 c.c. N/10 HCl = pH 0.993; 150 c.c. N/10 HCl = pH 0.9. (Chart plotted for me by J. F. McClendon.)

The plotted curve of acidity of 37 cases may be divided into several phases for study: (1) the ascension, usually the first hour, indicating the rapidity and intensity to a known stimulus; (2) the high point or acme, to note whether accelerated or retarded and whether abrupt or sustained; (3) the period of descent or decline and the possible secondary rise, also to note the character and modification of the food residues. The cases observed include chronic appendix and gall-bladder cases, before and after operation, hernia, carcinoma of the stomach, kidney stone, huge ovarian cyst, gastrojejunostomy, duodenal ulcer, prostatic hypertrophy and a few remote surgical conditions.

In Group I, 10 of 13 cases tend to a gradual rise, and at the end of the first hour have reached the maximum from which they usually

¹² Die Wasserstoff- Ionenconcentration des reinen Magensaften und ihre Beziehungen zur elektrischen Leitfähigkeit und zur titrimetrischen Acidität., Ztschr. Exper. Path. u. Therap., i, 431, 1905.

decline gradually. Three cases have a delayed rise. The average time juice could be obtained was two and a half hours, food being absent in the last fifteen minutes to three-quarters of an hour.

In Group II the curve either remains continuously high, or after a fall, undergoes a second rise, often higher than the primary one, near or at the end of digestion. In this first subdivision are 6 cases, 2 of which have a delayed rise; in the second subdivision are 12 cases, 1 having a delayed rise. The average time specimens could be obtained in the first division of Group II was two hours; in the second, two and three-quarter hours; in one-half of these cases food was present to the end.

In Group III, I have placed 5 cases of hypoacidity in which the acidity was less than pH = 4. Michaelis and Davidsohn place hypoacidity between 0.00041 and 0.0000001. Considerable peptic digestion takes place at pH = 4 and very rapid digestion at pH = 0.78 (Sorensen¹), so that digestion is retarded but not stopped until acidity is less than pH = 4. Dimethyl amino azobenzene does not react to an acidity less than about a pH of 4. In one of these cases the curve of acidity later, after removal of a gall-bladder with stones, rose to a normal acidity curve. The average time specimens could be obtained in this group was two and three-quarter hours.

Hypersecretion was demonstrated in gastric ulcer cases thirty years ago (Rubow). Recently it has been found not only in inflammatory conditions of the gall-bladder, appendix and in pelvic and other organs but in normal individuals. The vagus is considered one factor in producing this condition.

The values of pH before operation were taken in 8 cases with chronic appendicitis and a composite curve plotted (Fig. 3). The curve gradually rises to the end of the first one and a half hours, then falls slightly to the two-and-a-half-hour period, when it begins a secondary rise higher than the digestive rise, lasting to the end of collection. Two of these cases, having definite gastric disturbances, had early high acidity with an exaggerated secondary rise. Secretion in these chronic appendix cases was obtained for an average of two and three-quarter hours, but was obtained longer in the 2 cases with definite gastric symptoms. Food was present in 3 cases without symptoms to the end of observation, but in the others it was absent in the last three-quarters of an hour.

In addition to cases studied by the fractional method I ran a series of 14 clinical cases, comparing the secretion obtained one hour after an Ewald meal before and after operation for chronic appendicitis. I estimated the titration acidity in all of these cases, the total chlorides in 5 and determined the pH in 9 cases. There was some variation in the pH but no constant change after operation; therefore these results are not tabulated.

A composite curve of 7 cases of gall-bladder disease was plotted (Fig. 3). On account of variations in the individual acidity an irregular curve was formed. The curve rises rather suddenly in the

first three-quarter hour, then falls in an irregular wave to the two and a fourth hour period, when it begins its secondary rise higher than the digestive rise and retains it to the end of collection. Secretion was obtained in these cases an average of only a little over two

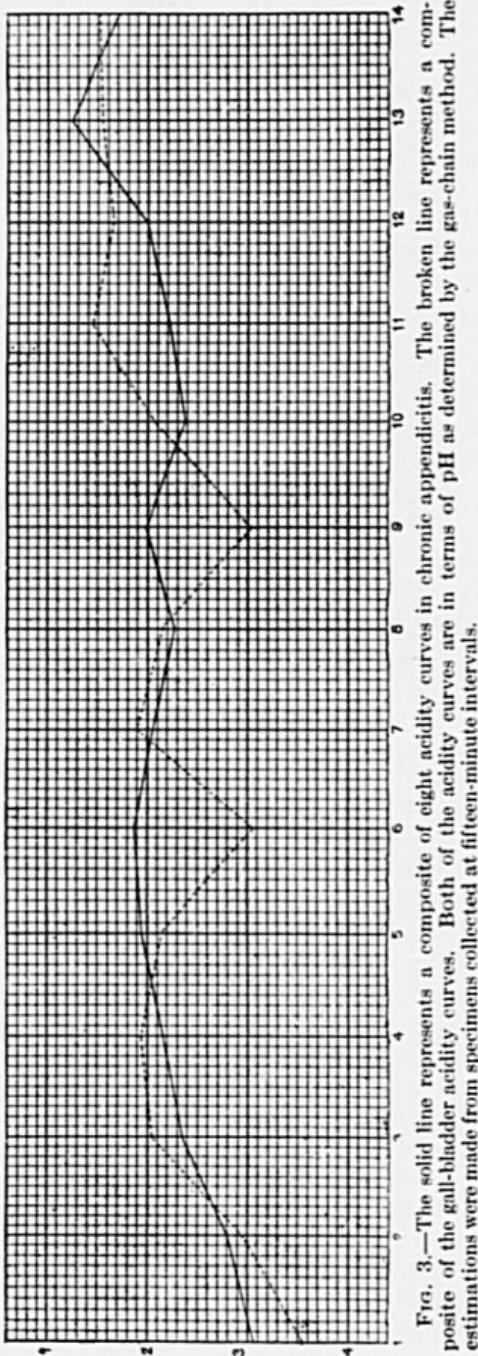


FIG. 3.—The solid line represents a composite of eight acidity curves in chronic appendicitis. Both of the acidity curves are in terms of pH as determined by the gas-chain method. The estimations were made from specimens collected at fifteen-minute intervals.

hours as compared to two and three-quarter hours in chronic appendix cases. Food was present in all of the specimens of secretion in only 1 case, in the others it was absent in the last fifteen minutes to three-quarter hour, while in the chronic appendix cases, though secretion was obtained three-quarters of an hour longer, food was present in 3 cases in all of the specimens. A two-year gastrojejunostomy is interesting from the high actual acidity with a secondary rise at the end, and the length of time secretion was obtained, three and a half hours. This rather increased length of time corresponds to other observations.

The gastric symptoms in chronic appendicitis may be caused primarily by a reflex spasm of the pylorus with secondary changes in secretion and acidity (Aaron,¹³ or, according to Fenwick¹⁴ and McGuire,¹⁵ the gastric secretions may be changed primarily, with the other changes secondary to the irritation of a hyperacidity. Fenwick believes the constant excess of free HCl gives rise to a spasmodic closure of the pylorus and also excites a violent gastritis with often interstitial hemorrhages. He believes that 12 per cent. of the cases of hypersecretion are due to disease of the appendix and that there is usually an increase both in quantity and acidity, when active irritation of the appendix is present. Paterson¹⁶ says that the appendix influences the gastric secretion but thinks it due to intestinal stasis rather than pyloric spasm. Ochsner¹⁷ says that there is undoubtedly a contraction of the ileocecal valve during an acute exacerbation of appendicitis. This in turn may give rise to a contraction of the duodenal and pyloric sphincter and in this way the normal passage of food from the stomach is impaired. Long and short, or local, reflex nerve paths to the stomach from the appendix and intestine have been demonstrated clinically and experimentally.¹⁸

¹³ Chronic Appendicitis, Pylorospasm and Duodenal Ulcer, a Preliminary Note, Jour. Am. Med. Assn., 1915, lxiv, 1845.

¹⁴ The Clinical Significance of Gastric Hypersecretion and its Connection with Latent Disease of the Appendix, Proc. Royal Soc. of Med., 1910, iii, 3, 177.

¹⁵ Tr. South. Surg. and Gynee. Assn., 1911, xxiii.

¹⁶ Appendicular Gastralgia, or the Appendix as a Cause of Gastric Symptoms, Proc. of Royal Soc. Med., 1910, iii, 3, 187.

¹⁷ Further Observations on the Anatomy of the Duodenum, AM. JOUR. MED. SC., 1906, cxxii, 1.

¹⁸ Opitz: Quoted by Kenefick, Med. Rec., 1915, lxxxviii, 355.

Cannon, W. B.: The Mechanical Factors of Digestion, 1911.

Cannon, W. B.: The Acid Control of the Pylorus, Am. Jour. Physiol., 1907, xx, 301.

Carlson, A. J.: Reflexes from the Intestinal Mucosa of the Stomach, Am. Jour. Physiol., 1914-1915, xxxvi, 191.

Alvarez, W. C.: The Motor Functions of the Intestine from a New Point of View, Jour. Am. Med. Assn., 1915, lxv, 388-394.

Irritability in Different Parts of the Stomach, 1916, xli, 321.

Keith, A.: An Account of Six Specimens of the Great Bowel Removed by Operation with Some Observations on the Motor Mechanism of the Colon, Brit. Jour. Surg., 1914-1915, ii, 576-599.

A New Theory of the Causation of Enterostasis, Lancet, London, 1915, ii, 371-375.

Barelay, A. E.: Radiological Studies of the Large Intestine, Brit. Jour. Surg., 1914-1915, ii, 638-652.

In my experimental observations I used the Pawlow pouch in order to obtain pure juice and found it of value in controlling operative and postoperative hemorrhage to run a buttonhole suture around the incised stomach wall before invagination. In order to lessen the tendency to breaking down of the mucosa between the main stomach and the pouch I invaginated the cut mucosa and submucosa as Keeton¹⁹ has suggested, not so that the closed edges of the two stomachs fell against each other, but so that they formed a cross

C
E.....A + B (Fig. 4).
D

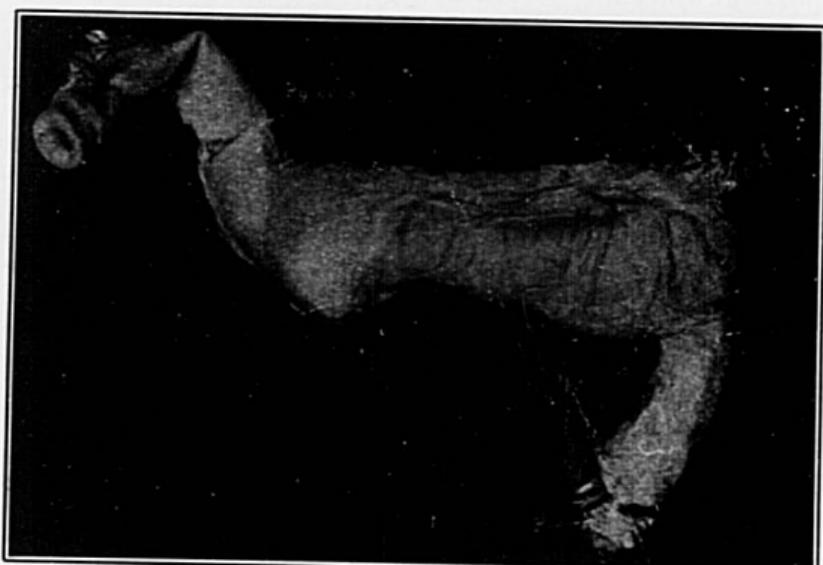


FIG. 4.—Photograph of Pawlow stomach in Exp. 5. Line E-A is the line of the incision through the stomach wall. From A to B the mucosa and submucosa have been cut and peeled back from the muscle after which the ends are invaginated along the line A-B and in the small stomach at right angles, C to D. The point D is located on the posterior wall opposite C. The pouch is completed by invaginating the stomach wall from A to F, leaving the small opening at F. The incision through the main stomach is completely closed from A to E.

In my experiments on dogs, lesions similar to chronic appendicitis with adhesions in the human were attempted. The cecum of the dog forms a rather long spirally twisted appendix. A stenosis of this cecum and adhesions to the colon, ileum and abdominal wall were produced.

ANIMAL EXPERIMENTS.

1. Pawlow pouch, January 17, 1917. Lesions of cecum, January 24, 1917. Slightly inflamed cecum removed and adhesions freed, January 29, 1917. Posterior gastrojejunostomy, March 5, 1917.

¹⁹ Am. Jour. Physiology, 1914, xxxiii, 25

Chloroformed, May 28, 1917. Gastro-enterostomy and Pawlow pouch results good.

2. Pawlow pouch, February 23, 1916. Collections. Lesions of cecum, April 10, 1916. Posterior gastrojejunostomy, May 8, 1916. Collections. Dog chloroformed, August 4, 1916. Necropsy: adhesions of cecum to colon, ileum and abdominal wall. Gastrojejunostomy opening patent.

3. Pawlow pouch, March 2, 1916. Lesions of cecum, March 27, 1916. Chloroformed, April 2, 1917. Wound infection. Definite inflammation of cecum.

4. Pawlow pouch, October 20, 1916. After collections, lesions of cecum, November 20, 1916. Collections. Reoperated. Chloroformed, November 25, 1916. Necropsy: acute inflammation of cecum and peritonitis.

5. Pawlow pouch, November 27, 1916. Collections. Lesion of cecum, December 13, 1916. Inflamed cecum removed, December 28, 1916, adhesions to ileum, colon and abdominal wall freed. Chloroformed, January 13, 1917.

6. Pawlow pouch, December 1, 1916. Collections made similarly to experiment 5 as a normal control.

7. Pawlow pouch, January 2, 1917. Lesions of cecum, January 12, 1917. Distemper. Chloroformed, January 20, 1917. Necropsy: adhesions and stenosis of cecum.

8. Pawlow pouch, January 5, 1917. Lesions of cecum, January 15, 1917. Cecum removed and adhesions freed, January 20, 1917. Chloroformed, February 28, 1917. Necropsy: small communication between the two stomachs. Ulcer at suture line.

9. Pawlow pouch, January 16, 1917. Lesions of cecum, January 24, 1917. Acutely inflamed cecum removed. Peritonitis. Chloroformed, January 30, 1917.

10. Pawlow pouch, January 19, 1917. Lesions of cecum, January 26, 1917. Collections. Cecum removed, January 29, 1917. Distemper. Chloroformed, February 13, 1917.

11. Pawlow pouch, November 24, 1916. Collections. Emaciation and vomiting. Chloroformed, December 13, 1916. Necropsy: marked hour-glass contraction of stomach. No other pathology.

The following contains animal experiments illustrative of my results, the first column under "pH," showing the acidity determined by the gas-chain method. Under "HCl" is placed the dimethyl amino azobenzene and under "Total" the phenolphthalein, acidity, determined by the titration method.

EXPERIMENT 1. One of four test meals from Pawlow stomach before lesions were produced. Test meal, 75 grams Hamburg and 2 grams peptone:

| Hr. | pH. | HCl. | Total. | C.e. |
|-----|------|------|--------|------|
| 1 | 1.19 | 94 | 106 | 6½ |
| 2 | 1.27 | 72 | 94 | 1½ |
| 3 | 3.43 | 0 | 54 | 1 |
| 4 | | ... | ... | ½ |

After production of stenosis of cecum and adhesions. One of five test meals:

| Hr. | pH. | HCl. | Total. | C.c. |
|-----|------|------|--------|------|
| 1 | 1.18 | 100 | 110 | 2½ |
| 2 | 1.18 | 90 | 104 | 1½ |
| 3 | 1.30 | 62 | 82 | 1 |
| 4 | | ... | ... | ½ |

After removing cecum and freeing adhesions. One of seven test meals, one to twenty-three days after operation:

| Hr. | pH. | HCl. | Total. | C.c. |
|-----|------|------|--------|------|
| 1 | 1.22 | 98 | 119 | 3 |
| 2 | 1.23 | 96 | 110 | 2½ |
| 3 | | 66 | 82 | 2 |
| 4 | | ... | ... | 1 |

One typical test meal from five tests, four to seventeen days after a gastrojejunostomy:

| Hr. | pH. | HCl. | Total. | C.c. |
|------|------|------|--------|------|
| 1 | 1.08 | 84 | 98 | 5 |
| 2 | 1.06 | 102 | 114 | 6 |
| 3, 4 | 1.01 | 106 | 118 | 8 |
| 5 | 1.03 | 98 | 110 | 4 |
| 6 | 1.01 | 100 | 112 | 2 |

EXPERIMENT 5. Four test meals made from Pawlow stomach. Illustrative meal after 75 grams Hamburg and 2 grams peptone:

| Hr. | pH. | HCl. | Total. | C.c. |
|----------------------|------|------|--------|------|
| Continuous secretion | 1.43 | 42 | 48 | 1 |
| 1 | 1.28 | 60 | 66 | 4 |
| 2 | 1.32 | 60 | 66 | 5 |
| 3 | 1.33 | 68 | 74 | 3½ |
| 4 | 1.60 | 80 | 86 | 3 |
| 5 | 1.52 | 54 | 60 | 3 |
| 6 | 1.50 | 64 | 68 | 2½ |
| 7 | 1.26 | 64 | 68 | 2 |
| 8 | 1.43 | 42 | 60 | 1½ |

After production of stenosis of cecum and adhesions. One of two test meals:

| Hr. | pH. | HCl. | Total. | C.c. |
|----------------------|------|------|--------|------|
| Continuous secretion | 1.35 | 78 | 86 | 4 |
| 1 | 1.20 | 74 | 84 | 20 |
| 2 | 1.20 | 64 | 72 | 14 |
| 3 | 1.15 | 90 | 100 | 10 |
| 4 | 1.14 | 84 | 88 | 8 |
| 5 | 1.33 | 82 | 88 | 8 |
| 6 | 1.36 | 80 | 82 | 7 |
| 7 | 1.25 | 80 | 88 | 10 |
| 8 | 1.26 | 76 | 82 | 9 |
| 9 | 1.52 | 80 | 86 | 6 |

Twelve days after removal of inflamed cecum. No appetite. Test meal given by tube:

| Hr. | pH. | HCl. | Total. | C.c. |
|----------------------|------|------|--------|------|
| Continuous secretion | 1.10 | 104 | 116 | 3 |
| 1 | 1.30 | 94 | 104 | 2½ |
| 2 | 1.30 | 98 | 106 | 3 |

EXPERIMENT 8. Average of five test meals from Pawlow stomach. Test meal, 75 grams Hamburg and 2 grams peptone:

| Hr. | pH. | HCl. | Total. | C.c. |
|-----|------|------|--------|------|
| 1 | 1.25 | 85 | 105 | 4½ |
| 2 | 1.18 | 92 | 109 | 6½ |
| 3 | 1.22 | 70 | 80 | 2 |
| 4 | 1.49 | 80 | 95 | 1½ |
| 5 | 1.65 | 50 | 78 | 2 |
| 6 | 1.18 | 95 | 103 | 1 |
| 7 | 1.20 | 92 | 100 | 1 |

After production of stenosis and adhesions of cecum. Average of four test meals:

| Hr. | pH. | HCl. | Total. | C.c. |
|-----|------|------|--------|------|
| 1 | 1.23 | 74 | 85 | 7 |
| 2 | 1.16 | 86 | 96 | 5 |
| 3 | 1.16 | 62 | 87 | 4½ |
| 4 | 1.18 | 74 | 89 | 1½ |
| 5 | | 28 | 54 | ½ |
| 6 | | 24 | 48 | ½ |

After removal of long cecum. Average of five test meals:

| Hr. | pH. | HCl. | Total. | C.c. |
|-----|------|------|--------|------|
| 1 | 1.17 | 116 | 128 | 8 |
| 2 | 1.15 | 121 | 133 | 10½ |
| 3 | 1.16 | 113 | 125 | 5½ |
| 4 | 1.16 | 90 | 106 | 3 |
| 5 | 1.20 | 108 | 118 | 1 |
| 6 | 1.20 | 108 | 118 | 1 |

EXPERIMENT 10. Average of three test meals from Pawlow stomach. Test meal, 75 grams Hamburg and 2 grams peptone:

| Hr. | pH. | HCl. | Total. | C.c. |
|-----|------|------|--------|------|
| 1 | 1.26 | 89 | 104 | 3 |
| 2 | 1.22 | 74 | 94 | 1½ |
| 3 | | ... | ... | ½ |
| 4 | 1.4 | 72 | 96 | ½ |

After production of stenosis of cecum and adhesions. Average of three test meals:

| Hr. | pH. | HCl. | Total. | C.c. |
|-----|------|------|--------|------|
| 1 | 1.14 | 102 | 116 | 4 |
| 2 | 1.13 | 107 | 122 | 1½ |
| 3 | 1.27 | 100 | 110 | 1 |
| 4 | 1.18 | 100 | 109 | 1 |
| 5 | 1.18 | 100 | 108 | 1 |

After removal of cecum and freeing of adhesions. Average of five test meals:

| Hr. | pH. | HCl. | Total. | C.c. |
|-----|------|------|--------|------|
| 1 | 1.12 | 118 | 130 | 3 |
| 2 | 1.14 | 116 | 124 | 4 |
| 3 | 1.12 | 116 | 119 | 1½ |
| 4 | 1.14 | 110 | 114 | 1 |
| 5 | 1.14 | 110 | 112 | ½ |

RESULTS OF EXPERIMENTS. In considering the results of the animal experiments, there was a definite increase in secretion in nearly one-half of the cases after lesions of the cecum were produced. In 1 case there was also an increase in continuous secretion; in the others there was a hypersecretion with a slightly longer period of flow. After removal of the "cecum" there was also a marked increase in quantity in 1 case and slight increase in another. The actual acidity after the lesions, as well as after the removal of the "cecum," was slightly higher than that ordinarily found corresponding to an increased flow of secretion. After gastrojejunostomy (Experiments 1 and 2) there was a decided increase in the rate of secretion in both cases as well as a prolonged flow of juice in 1 case. The acidity was slightly increased after gastrojejunostomy, associated with a more rapid rate of flow. After hour-glass contraction of the stomach in 3 cases there was no decrease of acidity, although frequently a decrease in such cases has been found clinically. There was a slightly increased rate of flow in some instances, in others a decreased rate, associated with emaciation and vomiting. One case, Experiment 11, is listed.

SUMMARY OF CLINICAL AND EXPERIMENTAL WORK. In a group of surgical cases, it has been shown that the curve of acidity may rise higher at the end of digestion than it has during the digestive rise. After certain surgical lesions have been made upon the gastrointestinal tract of animals, it is demonstrated that there is a slight increase in acidity in the secreted gastric juice; in some reflex manner, however, lesions of the cecum or other changes may cause an increased rate of flow. There must be some disturbance of the factors concerned in neutralization of the acidity associated with the factors controlling the discharge of chyme through the pylorus. The contour of the curve of acidity must be influenced by the food and fluids present, the intensity and duration of the appetite and food secretion, the emptying time of the stomach, the regurgitation of alkaline and duodenal contents, the gastric mucus and the saliva.

CONCLUSIONS. 1. The clinical study of the gastric secretion must be done by examination of specimens taken at short intervals during digestion. Examination of a single specimen for comparative purposes is inexact and inadequate.

2. There is often a persisting high acid secretion after the digestive period. The digestive acidity may fall with a secondary late rise. Frequently surgical cases have this type of curve.

3. Clinically, the pH derived from the titration method is uniformly higher than the actual acidity as determined by the gas-chain method.

4. In animal experiments, after production of chronic inflammation of the long appendix of the cecum, with adhesions, after the removal of this appendix, after gastrojejunostomy and hour-glass stomach, no definite increase in actual acidity of pure gastric

juice was observed. There was an increased rate of flow and a prolonged flow in some cases.

5. The presence of increased acidity in the stomach contents in these surgical conditions must therefore be explained by factors noted in the summary above and not to an actual increased acidity of the gastric juice.

**NOTES ON THE PROGNOSTIC VALUE OF PSYCHOMETRIC
TESTS AS COMPARED WITH CLINICAL SIGNS IN
EPILEPSY.¹**

By L. PIERCE CLARK, M.D.,

CHIEF CONSULTING NEUROLOGIST AND PSYCHIATRIST TO THE RANDALL'S ISLAND
INSTITUTIONS,

AND

ALEXANDER MILEAU, M.D.,

ASSISTANT PHYSICIAN, RANDALL'S ISLAND INSTITUTIONS, NEW YORK.

FROM data now at hand it is obvious that the diagnosis and prognosis of many cases of essential epilepsy rest in no small degree upon accurate analysis of the so-called mental stigma of the disorder, the epileptic constitution, as well as the modifiability of the latter under appropriate training treatment. Likewise the course of the disorder is to be judged by the presence or absence of deterioration and its degree.² As regards deterioration, in the past we have been content for the most part to indicate it purely on the basis of a clinical estimate of behavior, ability to do work and like data. However, it now seems opportune for us to call to our aid a more precise method to determine the initiation of deterioration not only for prognostic reasons but also that one may early determine the efficacy of any definite plan of training treatment. The psychometric work of Eberschweiler and Jung and, more especially, the proofing test of Hahn, are suggestions in point. Although a large number of studies of this psychological character has appeared, Hahn³ was able, in 1913, to check up some of his work on the same clinical material studied in 1903 and 1908. The time interval between the two sets of studies were from two to four years. As is natural the findings in his studies had to exclude the mental infirmity due simply to an arrest in growth and development as well as a proper allowance made for the wide variations of the primary endowment in such individuals. Hahn employed a

¹ Delivered as a part of the Symposium on Epilepsy and Allied States, Randall's Island, June 4, 1917.

² Clark: Clinical Studies in Epilepsy, Psychiatric Bull., January, 1916, to January, 1917.

³ Association Studies in Youthful Epileptics, Archiv f. Psychiatrie, 1913, iii, 1078.
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